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Multifactorial Hypercalcemia

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Hypercalcemia (ca) has been described in many patients (pt) with granulomatous diseases. We present a pt with persistent hyperca from a combination of primary hyperparathyroidism (PHPT) and elevated 1,25 dihydroxyvitamin (OH2) D levels from reactivated tuberculosis (TB). An 82-year-old woman with a past medical history of Waldenström's macroglobulinemia and TB presented to hospital for imbalance and fatigue. Examination was unremarkable. Labs were notable for newly elevated ca of 14.8 mg/dl. After 200 mg of subcutaneous calcitonin on day 1, ca levels transiently improved but worsened by day 3. Calcitonin 200 mg twice daily was given for 2 days. Cinacalcet 60 mg daily was started for persistently elevated ca, which was then increased to twice daily. Two days later, cinacalcet was increased to 90 mg twice daily. Laboratory work-up revealed parathyroid hormone (PTH) to be inappropriately normal (45 pg/ml with concurrent ca levels of 14.8 mg/dl). Serum vitamin D levels were normal. Sestamibi scan showed bilateral PT adenoma for which she underwent parathyroidectomy. Pathology showed PT hyperplasia. Post-surgical PTH levels were down to 15 pg/ml but calcium levels remained elevated at 14 mg/dl, for which pamidronate 30 mg was given. Further work up showed elevated 1,25 OH2 D levels at 278 pg/ml and repeat levels were 261 pg/ml. Dexamethasone 6 mg daily was started while the pt was worked up for etiologies of elevated 1,25 OH2 D levels, with no improvement. PTHrP, SPEP and UPEP with IFE, quantiferon, and thyroid functions tests were normal. Radiology was unremarkable. AntiTB, and antifungal agents were empirically started. The latter was discontinued upon negative cultures.1,25 OH2 D levels improved with antiTB drugs continuation. Dexamethasone was tapered and pt completed antiTB treatment with normalization of ca levels (despite negative TB culture which resulted after the 1,25 OH2 D levels improved). PHPT is the most common cause of hyperca whereas malignancy is the most common cause of non PTH mediated hyperca in hospitalized patients. In our pt, ca remained elevated despite appropriate treatment of PHPT, suggesting other causes for hyperca. Although our pt fit the demographic for neoplasia, elevated 1,25 OH2 D levels indicated non-neoplastic etiology. Improvement in 1,25 OH2 D and ca levels following initiation of antiTB drugs supported mycobacterial infection reactivation as the most likely etiology, especially given pt's history of TB. In pts with granulomatous diseases, activated macrophages convert 25 OH D to 1,25 OH2 D without the usual regulation by PTH, ca and phosphorous that control renal 1-alpha hydroxylase activity. Common etiologies of 1,25 OH2 D include sarcoidosis, mycobacterial infections, and lymphoma. The presence of concurrent PTH and non PTH mediated etiology of hyperca makes this case an interesting one and showcases that multifactorial etiologies of hyperca can coexist.

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